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Scab of Wheat:
Prospects for Control

Scab of wheat, also called Fusarium head blight, is a destructive disease in the humid and semihumid wheat-growing areas of the world (40). J. C. Arthur documented an outbreak of wheat scab in Indiana that occurred in 1890 and observed that one field that had a reasonable expectation of a yield in the range of 2,350–2,690 kg/ha (35–40 bu/ac) in fact yielded only 538 kg/ha (8 bu/ac) because of the disease (3). Scab incidence in that epidemic ranged from 25 to 75%. There have been several recent outbreaks of scab in the eastern soft red winter wheat region of the United States (53). Moderately widespread scab epidemics were reported about once every 9 years in southwestern Ontario, Canada, from 1927 to 1980 (49). In the People's Republic of China, wheat scab can be found in two-thirds of the provinces, where it afflicts more than 7 million hectares of wheat (56). Epidemics are most severe in the middle and lower reaches of the Yangtze Valley, coastal areas of southern China, and the eastern part of Heilongjiang province (70). Scab is one of the most important wheat diseases in those areas. Wheat scab is also becoming a greater threat to wheat production in many other countries (29,39,44,46,49). In 1993, scab was severe in the spring wheat areas of the northern United States and in the province of Manitoba, Canada.

Wheat scab can greatly reduce grain yield and quality, and may indirectly affect animal production. Direct yield reduction results from shriveled grains, which may be light enough to be expelled from the combine with the chaff. Diseased kernels that are not eliminated with the chaff reduce test weight because they

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are light and shriveled. Scab causes indirect loss by reducing seed germination and causing seedling blight and poor stand (53). Scab is often epidemic over large areas but will show local variation in severity because of crop rotation, date of anthesis, and microclimatic influences. In a severely affected field, virtually every head of wheat can show symptoms (40). In China, the incidence of scabbed heads may range from 50 to 100%, with yield loss as high as 20–40% in years of severe epidemics. It was estimated that over 7 million hectares of wheat had scab and more than 1 million tons of wheat were lost in a severe epidemic year (56). In an epidemic in 1986, grain samples from 43 of 44 counties in Indiana had scab, and scabbed kernels represented an average of 2.9% by weight of each combine-harvested grain sample. Kernel infection averaged 23% (53). In the Atlantic provinces of Canada, scab was particularly important in 1980, causing 30–70% yield loss in spring wheat (27).

Scab can cause additional loss for agriculture because of the potent mycotoxins produced by the fungus. The two most important mycotoxins produced by *Fusarium graminearum* Schwabe (teleomorph = *Gibberella zeae* (Schwein.) Petch or *G. saubinetii*) are the estrogenic toxin zearalenone and the trichothecene deoxynivalenol (DON), a vomitoxin (46, 49,53). In 1986, Clear and Abramson (15) reported that in Manitoba, 12.6 and 9.6 ppm of DON were detected in the grain samples of hard red spring wheat and amber durum, respectively. A survey of wheat grain in Indiana in 1986 revealed DON in 88% of samples, with a mean concentration of 0.6 ppm. Nine percent of the samples had more than 2 ppm DON (53). Other investigations showed that DON was found at high levels in scabbed wheat seeds (18,46). These mycotoxins have been tentatively linked with livestock toxicoses or feed refusal. Grain with one or both toxins may be graded down or rejected entirely in commerce (53).

Pathogens

Many species of *Fusarium* can cause wheat scab, and the symptoms caused by different species are almost the same. *F. graminearum* is the principal pathogen responsible for head blight in many countries (15,40,49,56,59). Other related species such as *F. culmorum, F. avenaceum, Microdochium nivale, F. moniliforme, F. oxysporum,* and *F. poae* may also contribute to the head blight complex but are generally less important than *F. graminearum* (12,46,49,56,58,59).

F. graminearum can survive as mycelium, ascospores, macroconidia, and chlamydospores (34,39,49,59). Microconidia are not produced. Macroconidia, mycelia, and chlamydospores are formed by the *F. graminearum* stage; ascospores are the propagules of the sexual stage. In soil, macroconidia or mycelium may be transformed into chlamydospores (34,49).

Cultural variation is a common phenomenon for *Fusarium* species. Oswald (35) classified isolates into two major types, I and II, based on sporulation and growth form. Type I, usually found in nature, has abundant aerial mycelium, and the conidia are in sporodochia. Type II usually occurs as variants in culture and has appressed and slimy growth due to the production of numerous conidia in pionnotes. Single-spore isolation after subculturing for several generations can produce many variants, some apparently not even of the same species as their parents (35).

Purss (37) observed that isolates of *F. graminearum* from corn or crowns of wheat could produce severe head blight but that crown rot was produced only by isolates from infected crowns. He considered this phenomenon to be pathogenic specialization in *F. graminearum* and pointed out that the severe crown rot syndrome in Queensland was caused by a distinct pathogenic race. The two natural populations of *F. graminearum* were further distinguished as group 1 and group 2 by their life cycles and ecological

requirements (16). Group 1 isolates were normally associated with crown rot, did not form perithecia in culture, rarely produced the *Gibberella* stage in nature, and could cause scab during abnormally wet weather in the semiarid regions. Group 2 isolates were associated with head scab in cereals and stalk rot and ear rot of corn in humid climates, and could produce the *Gibberella* stage in nature (16,37).

Isolates of *F. graminearum* differ in pathogenicity, but probably not in a race-specific manner (5,52,54). Although a significant interaction between cultivars and isolates has been shown (52,54), there is no evidence for stable pathogen races (5,28,29,54). Based on the test of cultivar resistance to different species of *Fusarium,* Mesterhazy (29) concluded that cultivar resistance to certain strains of *F. graminearum,* as well as to other species of *Fusarium,* was not strain or species specific. A mixture of highly pathogenic isolates was recommended as inoculum for evaluation of resistance (56).

Sources of Inoculum

F. graminearum survives between wheat crops in living or dead host tissues. Ascospores, macroconidia, chlamydospores, and hyphal fragments all can serve as inoculum (34,39,69). Ascospores and macroconidia may be the principal inoculum that initiates an epidemic of scab, because aerial dispersal is necessary for the fungus to reach the infection site (49).

The source of primary inoculum for scab varies with cropping systems in different parts of the world, but in all cases crop residues on the soil surface are the most important source of inoculum. Debris of wheat, corn, or rice can be the principal reservoir of the fungus (1,4, 49,58,69). In southern China, where wheat is planted after rice, rice stubble is a major source of inoculum for wheat scab (69). Perithecia may occur on 99% of rice stubble in late December. In Pennsylvania, abundant mature perithecia of *G. zeae* were found on corn and wheat debris from April to December (4). Wheat planted after corn often has significantly more scab than wheat planted after other crops (50,51). Reduced tillage for soil conservation increases the amount of inoculum that can infect wheat (50,51).

Scab Development

Airborne ascospores are deposited on or inside wheat spikelets, where they germinate and initiate infection. The fungus first infects the extruded anthers and then ramifies throughout the developing caryopsis, floral bracts, and rachis. The severity of infection is correlated with the percentage of retained anthers (36). The fungus may also infect by direct penetration of the glume, palea, or rachilla (12). Soon after infection, dark brown, water-soaked spots appear on the glumes of infected spikelets. Later, entire spikelets become blighted (Fig. 1). Under favorable conditions, the fungus extends through the rachis, externally or internally, and infects other spikelets (12). If the weather is warm and humid, conspicuous aerial mycelium spreads externally from the spikelets originally infected to adjacent spikelets. If the fungus spreads internally, brownish chlorotic symptoms extend down into the culm and up to the top of spike, and as a result the entire spike may die and turn the color of straw (Fig. 2). Visible pink mold appears on the spike when it is humid. Later in the season, perithecia of *G. zeae* may appear as raised purplish black spots (59). Infected florets often fail to produce grain, or the grain they produce is poorly filled (Fig. 3).

Wheat is generally most susceptible to infection at the flowering stage (2,3,36), and susceptibility declines at later stages of caryopsis development. Some cultivars, however, may be most susceptible at the milk or soft dough stages (1,40). Head infection in nature can occur anytime after the beginning of flowering, when temperature and moisture are favorable. Anthesis may be the period of greatest susceptibility, because the high levels of choline and betaine produced in the extruded anthers stimulate the growth of *F. graminearum* and promote the infection of wheat spikes by the pathogen (47,48). Because the main period of vulnerability of wheat to infection is during anthesis, the fungus is generally limited to one infection cycle per season. The abundance of primary inoculum and weather conditions during anthesis determine the severity of Fusarium head blight. Nutrient stress may increase the plant's susceptibility to infection. Weeds in a field could affect disease by increasing water or nutrient stress on the wheat, by modifying the crop moisture environment, or by serving as a host of *F. graminearum* during the off season (51).

The incubation period is as short as 2–3 days in the greenhouse or laboratory (5,8,56), and 4–5 days in the field (61). Primary infection may occur on several florets in the field. The dark brown symptom usually extends into the rachis, even down into the stem tissue. The pathogen can be found in the plant tissues with the dark brown symptom. Mycelium invades parenchymatous tissue as well as vascular tissue (40). The clogging of vascular tissue in the rachis can cause the head to ripen prematurely, so that even grains not directly infected will be shriveled because of a shortage of water and nutrients (40). If heads are extensively invaded at a very early stage, kernels may fail to develop entirely.

Andersen (2) found that the optimum temperature for infection and head blight development was 25 C. Little or no infection occurred at 15 C. Incidence increases as temperature increases from 20 to 30 C. The moist period required for infection ranges from 36 to 72 hours, depending on temperature and plant growth stage (2). Wet periods during the flowering stage are important to infection. The duration of the wet period required for heavy infection depends on temperature; it is shortest at 25 C (2,36). One overnight (16 hours) moist period may be enough for infection on some cultivars in the greenhouse (5).

Control Measures

Because of the ubiquitous nature and wide host range of *F. graminearum* and other *Fusarium* spp. capable of causing scab, adequate control by crop rotation alone is not possible (27,39). However, crop rotation, coupled with plowing to bury infested crop residues and weed hosts, can be effective. Appropriate methods of land preparation, rotation with appropriate noncereal crops, good crop husbandry, timely harvest, and proper storage all help reduce disease by reducing primary inoculum and the likelihood of extensive infection (27, 41,51). Of these various cultural control practices, elimination of sources of primary inoculum has been very important. The adoption of minimum tillage for soil conservation greatly reduces options for this disease management technique.

Seed-treatment fungicides reduce the spread of seed-borne inoculum and increase seedling vigor (27,50) but have little effect on head blight because of the large amount of inoculum that can infect the wheat head directly later in the growing season. Although a foliar application of fungicide at anthesis might provide some protection, there are few fungicides available for application this late in the season (11,42). Cost of treatment and the difficulty of determining the optimum time of application also make this means of control less attractive to farmers. Even if a fungicide reduces direct yield loss, it may not reduce mycotoxin contamination to a tolerable level (27).

Cultivar Resistance

Arthur (3) was the first to note differences in susceptibility to scab among wheat cultivars. Considerable effort since then has been devoted to finding sources of resistance that can be used in breeding programs (8,17,23,24,57). Christensen et al (14) tested 350 accessions of *Triticum* spp. and found marked differences in their susceptibility. Hanson et al (17) summarized the results of evaluations for resistance on hundreds of cultivars and thousands of hybrids and selections of *Triticum* spp. from 1935 to 1950. Most were susceptible, but a few were relatively resistant. Cultivars Progress and Haynes

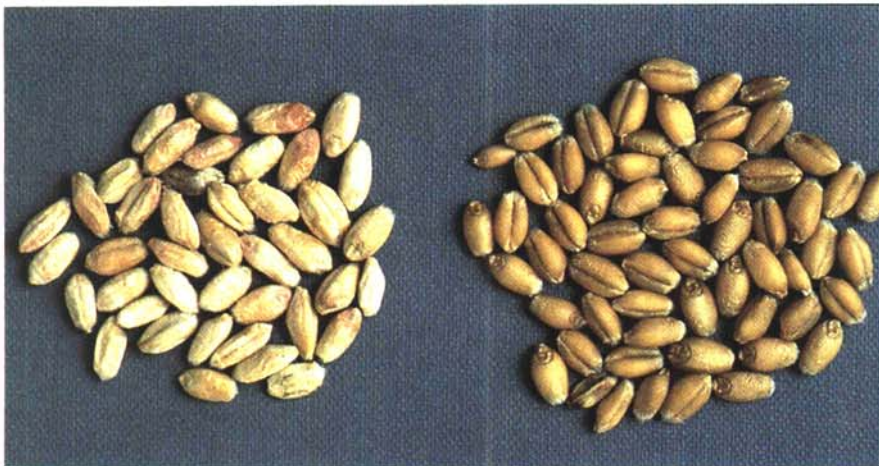Fig. 1. Wheat spikes infected by *Fusarium graminearum* in the field.

Fig. 2. Scab-resistant wheat cultivar Ning 7840 (left) and scab-susceptible wheat cultivar Clark (right). The spikes were inoculated in the greenhouse 3 weeks before the photograph was taken by injecting 1,000 conidia of *Fusarium graminearum* into a central spikelet. On Ning 7840, only the inoculated spikelet developed symptoms, while on Clark the entire spike became scabbed.

Fig. 3. Wheat grain scabbed as a result of infection by *Fusarium graminearum* (left) and healthy grain (right). The scabbed grain was extracted from a sample that had been harvested with a combine.

Bluestem were most resistant, but their low level of resistance discouraged breeders from working with them further.

Considerable progress in the search for resistance has been made in China in recent years (56,57,60,63,67,70). More than 17,000 common wheat cultivars or advanced lines were tested in the field and laboratory at many institutes in the southern winter wheat region from 1977 to 1983 (57). Only 32 of these showed a high degree of resistance; most cultivars were very susceptible. Resistant cultivars were found mainly among those grown in the Yangtze Valley, where scab epidemics occur frequently. Most of the 32 resistant cultivars were tall land races that had small heads, matured late, and had other undesirable agronomic traits. Because of their low yield potential, these cultivars could not be used directly in commercial production. Some of them were used as parents in breeding programs, but the resistance was difficult

to incorporate into elite lines (25,57). Sumai 3 was reported to have high combining ability for both scab resistance and yield traits, and has been widely used in wheat breeding programs with some success (10,25,57,70).

In the Jiangsu Academy of Agricultural Sciences of China, 389 new lines from Jiangsu and 414 from other areas were tested for scab resistance in 1987 and 1988 (8). Only 3% of the lines tested were rated resistant, 9% were moderately resistant, and 22% were moderately susceptible. Most of the resistant lines (about 75%) were derived from Sumai 3. Some of these lines had relatively stable resistance, similar to Sumai 3, and improved agronomic traits compared to their resistant parent. The moderate yielding and highly disease resistant wheat cultivars Ning 7840 and Ning 8026, which are derived from Sumai 3, have excellent resistance to scab (Fig. 2), as well as some resistance to leaf rust, stem

rust, and powdery mildew (56,67). Ning 8623, 8633, 8675, 8641, and some other lines possess moderate resistance to scab and have higher yield potential (4,850–6,100 kg/ha), shorter stature, higher test weight, and better processing quality than Sumai 3 (8). Some moderately susceptible cultivars, such as Yangmai 3, Yangmai 4, and Yangmai 5, have high yield potential and have been widely adopted for commercial production. Although these cultivars had a high incidence of scab in an epidemic year, the loss of yield was far less than in susceptible cultivars.

Evaluation of Resistance

Resistance in wheat to scab is of at least two types: resistance to primary infection and resistance to spread of hyphae within a wheat spike (40). To distinguish these types of resistance, different inoculation techniques were developed. Scattering scabby wheat kernels in the field to increase primary (ascospore) inoculum concentration was adopted by wheat breeders because of its low cost and convenience (56,67). Infection occurs naturally. The technique can be used to identify both types of resistance and to screen segregating populations on a large scale. However, many variables not directly related to host resistance affect primary infection. Plant height, moisture conditions, and the degree to which florets open may influence the amount of infection in any given test, and many susceptible plants may escape infection. For this reason, scattering infested seeds in field plots may not be suitable for an accurate test of resistance to scab spread within a spike. To ensure that each plant receives a similar amount of inoculum and to reduce the chance that a plant escapes infection, the method of placing spores

into a central spikelet was developed to detect cultivar differences in susceptibility to the spread of fungus within a spike (55). Because resistance to the spread of fungus is a more stable character than is resistance to primary infection (5,9,56), the technique is useful for studying the inheritance of scab resistance and for screening germ plasm and advanced breeding lines for scab resistance.

Classification of the scab reaction phenotype is complicated by the existence of the two types of resistance and their quantitative nature. The percentage of infected spikes (incidence) is used to measure resistance to primary infection, but it does not provide information on resistance to spread of the pathogen within a spike. To evaluate resistance of this latter type, two major rating systems are commonly used. Wang et al (56) proposed a four-grade rating method based on whether or not the fungus spread in the rachis from the site of initial infection. Grade 1 indicates symptoms only in the inoculated spikelet, grade 2 indicates fungal spread to the rachis but not to noninoculated spikelets, grade 3 indicates fungal spread to one noninoculated spikelet via the rachis, and grade 4 indicates spread of infection to two or more noninoculated spikelets. On this scale, grade means of 1–1.9 represent resistance, 2–2.9 moderate resistance, and 3–4 susceptibility. Xu and Fan (62) modified this scale by defining the spread of infection from one noninoculated spikelet to half the spike as grade 4 and spread to more than half of the spike as grade 5. Another rating system is based on the percentage of scabbed spikelets. In this system, resistance is evaluated by comparing test lines with control cultivars which have different levels of known resistance. It can distinguish susceptible genotypes from moderately susceptible genotypes (7,10). When the data are collected several times during the development of scab from an initial inoculation of a single floret, area under the disease progress curve can be calculated based on the proportion of scabbed spikelets (5). This summary statistic reflects the overall performance of a cultivar during the disease cycle. It allows relatively precise characterization of a cultivar's resistance, and it has been used in genetic studies (6). Under conditions of natural infection, both disease incidence and severity data are needed to measure the two types of resistance.

Since the 1980s, resistance to the formation of mycotoxins has received increased attention. Acetylated secondary metabolites of *F. graminearum* are deacetylated by plant enzymes. For instance, deoxynivalenol can be formed by wheat and corn cells from 3- or 15-acetyldeoxynivalenol produced by the fungus (31,32). The resistant cultivar Frontana may be a less suitable medium for accumulation of deoxynivalenol than

other cultivars because of the presence of enzymes that degrade deoxynivalenol (30). This characteristic of some wheat cultivars is regarded as a third type of resistance (31). The etiolated coleoptile bioassay was designed to determine the biological activity of metabolites of *F. graminearum* and the response of wheat cultivars to such compounds (56). However, lack of correlation between scab severity and the concentration of DON suggests that resistance to scab and resistance to mycotoxin accumulation may not be controlled by the same genes (27). Our results also indicated a poor correlation between coleoptile elongation and head scab resistance (G. Bai and G. Shaner, *unpublished*).

Inheritance of Resistance

Christensen et al (14) first showed that resistance to scab was an inherited characteristic and observed transgressive resistance among progenies from the cross of Marquis by Preston. Since the 1970s, scab epidemics have been more frequent throughout the world, and there has been correspondingly greater interest in the inheritance of scab resistance. Many investigators consider scab resistance to be quantitatively inherited and controlled by many minor genes (13,22, 44,60,64,65), but others provide evidence of oligogenic control (7,10,21,33).

Use of generation mean analysis to study scab resistance in crosses between resistant and susceptible cultivars indicates that resistance in some cultivars is mainly controlled by additive genetic effects, but nonadditive effects might also be significant (6,9,13,45). Within the nonadditive components, dominance appears to be the most important (10,13, 44). Epistatic effects were considered significant in some studies (6,45) but not in another (70). Heritabilities are usually high (9,13,22), but there are exceptions (66).

Monosomic analysis showed that many resistant cultivars possess genes for both resistance and susceptibility (65). Resistance genes were assigned to several chromosomes. Sumai 3 has scab resistance genes on chromosomes 1B, 2A, 5A, 6D, and 7D (64). Wangshuibai has resistance genes on chromosomes 4A, 5A, 7A, 7B, and 4D (22). The genes for resistance to scab in cultivar PHJZM are located on chromosomes 6D, 7A, 3B, 5B, and 6B (65). The moderately susceptible cultivar HHDTB has genes for resistance on chromosomes 5D, 1B, 7B, and 4D. In cultivar YGFZ, resistance genes are located on chromosomes 3A and 4D (65). Because the genes for resistance in different cultivars appear to be on different chromosomes, crosses between these cultivars may yield transgressive progenies with greater resistance than any of the parents.

Li and Yu (21) suggested that disease resistance could be measured in five

ways, namely: incubation period, time required for disease to spread from the infection site to the rachis, daily rate of scab progress before and after symptoms reach the rachis, and severity. They concluded that the spread of the fungus to the rachis was an important criterion in disease rating. The daily rates of scab progress before and after it reached the rachis were independent of each other. Resistance at different stages of scab development might be controlled by different genes. In cultivar WZHHS, genes on chromosomes 1B, 2A, 3D, 4B, 6A, 6D, 6B, 7B, and 7D affected the incubation period; genes on 3D, 6A, and 7D were responsible for spread of the fungus from the inoculated spikelet to the rachis; and genes on 2A, 3D, 4D, 5B, 6B, and 7D were responsible for spread of fungus to the entire spike. Genes on chromosomes 3D and 7D might control the rate of spread throughout three stages of disease development. The accumulation of resistance genes that operate at different stages of disease development may enhance the overall resistance of a cultivar.

Although resistance to scab is a complicated quantitative trait, resistance to spread within a spike is the main component of resistance and may be controlled by a few major genes (7,10, 25,33). Nakagawa (33) reported in 1955 that three pairs of epistatic factors might condition scab resistance in some wheat cultivars. Bai and Xiao (7) tested six cultivars and their F1, F2, F3, and backcross progenies in the field by spraying conidia on spikes during anthesis. Segregation of the F2 showed a continuous distribution, but two peaks coincided with both the resistant and the susceptible parents. The ratios of resistant to susceptible plants suggested segregation of two or three genes. Various resistant parents appeared to contain from one to three genes. It was hypothesized that the cultivars with one gene would be moderately resistant or moderately susceptible, cultivars with two genes would be either resistant or highly resistant, and cultivars with three genes would be highly resistant. The degree of resistance conferred by one or two genes would be more influenced by environment and genetic background than would the resistance conferred by three genes. Major genes at different loci may differ in their effects and may show complementation. Minor genes may also function as modifiers of the major genes, as reported in resistance to stripe rust (7,20). Similar results were obtained for resistance to the spread of scab in the spike following injection of spores into a central spikelet (10).

Breeding Strategies

The most resistant cultivars currently available were selected from the progeny of crosses between moderately suscepti-

ble parents or from crosses between a resistant cultivar and a susceptible but agronomically superior cultivar. Sumai 3 was selected from a cross of two moderately susceptible cultivars, Taiwanmai and Funo. Because of its high general combining ability for scab resistance and some other traits, Sumai 3 was used as a resistant parent with some success in China (8,9). Ning 7840 and several other resistant cultivars are derivatives of Sumai 3. Another indigenous cultivar from China, Wanshuibai, which is more resistant than Sumai 3, was also used as a resistant parent; but no resistant cultivars were selected from its progenies because of low combining ability for agronomic traits (9). Because highly resistant cultivars usually have undesirable agronomic traits that cannot be entirely eliminated (8,9,24), we may achieve more success by selecting transgressive segregants from crosses of moderately resistant cultivars with better agronomic characters (24,57).

To distinguish between genetic and environmental variance, an adequate environment for disease development is required. In China, some successful breeding programs are equipped to provide light overhead irrigation in breeding nurseries to simulate rainfall after anthesis. To select for resistance in early segregating generations, large test populations are inoculated by scattering scabbed wheat kernels in the field before plants reach the booting stage, then spraying plants with water during and after anthesis. To screen more advanced generations of near-homozygous progenies, spores are injected into a central spikelet under more controlled conditions. Lines that pass this selection test are sent to different scab epidemic areas for evaluation of resistance and yield potential.

To select for quantitative resistance and other characters, Wu (60) proposed a modified recurrent selection method. With the aid of a dominant male-sterile gene, $Ta1$, gene pools were developed by crossing several parents followed by recurrent selection (phenotypic mass selection and progeny selection). A long-term gene pool was created to maintain and accumulate genetic diversity for scab resistance, as well as to provide new germ plasm from which high-yielding cultivars resistant to scab under lower selection pressure could be selected. In the short-term gene pool, high selection pressure was applied to each cycle of recurrent selection to develop resistant cultivars as quickly as possible. To enhance the resistance gene pool, susceptible plants are regularly discarded, and new resistant germ plasm is added to accumulate diverse resistant genes.

When adequate resistance to a disease cannot be found within *Triticum aestivum,* breeders often turn to wild relatives as a source of resistance genes.

In an early search for resistance, only a few lines appeared to have resistance to scab (14). In China, 5,831 accessions of alien species were tested for scab resistance in several research institutes from 1977 to 1983, and no highly resistant materials were found (57). Liu et al (23) reported that *Roegneria ciliaris, R. kamoji,* and *Elymus giganteus* had resistance to scab; and some resistant plants were obtained from the segregating progenies of the crosses of those species with wheat cultivars. Recently, *Agropyron ciliare* and *A. repens* have also been reported to be resistant to infection (70). However, the resistance found in alien species may not surpass the resistance available in wheat cultivars (24).

Scab is a head disease, and direct phenotypic assessment of resistance must be delayed until the main culm of a plant reaches the anthesis stage. This precludes use of the tested spike as a parent in the same generation in which the resistance phenotype is assessed. If the tested plant produces tillers, these may be used for making crosses; but in the greenhouse, fertile tillers may not always form. Moreover, even in cases in which scab resistance appears to be controlled by major genes, there is a nongenetic component of phenotype that can make classification of individual plants in segregating generations uncertain. For these reasons, scab resistance breeding would be greatly facilitated by marker-assisted selection.

Morphological characteristics such as plant height, spikelet density, spike morphology, and awnedness have received attention as possible markers (13,22,43,45). However, the association between these traits and resistance is not consistent (26,65,68). DNA-based markers may provide a powerful tool for improved selection in plant breeding programs (38). The problems in phenotypic estimation of scab resistance associated with environmental effects or quantitative inheritance can be eliminated by the use of such markers. These approaches are expected to have four advantages in selection for wheat scab resistance over conventional methods. First, as DNA is evaluated directly for sequence polymorphism, environmental influences upon the expression of the polymorphism are removed as a source of confusion in the selection process. Second, since genomic DNA should remain constant at all stages of growth and under different environments, assays conducted at the seedling stage may permit the breeder to make selections well before crosses must be made. Third, selection intensity can be increased greatly, and selections can be made with confidence in early generations. Finally, a small amount of tissue can be assayed rapidly without destroying the original plant, which allows selection of specific

genotypes from among a complex group of individuals (19). This method should make it possible to combine resistance genes from different sources to improve scab resistance and other traits.

Although progress has been made in finding and analyzing scab resistance in wheat, most cultivars in current production are susceptible. Combining desired agronomic characters with a high degree of resistance is still difficult. Most resistant cultivars in China were derived from Sumai 3, and this same source of resistance is now being used in breeding programs in other parts of the world. This could create genetic homogeneity for scab resistance, with consequent genetic vulnerability if the fungus is able to adapt to this single source of resistance. Finding new resistant germ plasm and developing techniques for combining resistance genes are important tasks for wheat breeders. A rapid, efficient, and accurate technique for identifying resistance in segregating material needs to be developed so that large populations can be screened. The development of selectable markers, especially RAPD markers, may provide this technique; but first, considerable work is needed to find and characterize sources of resistance and to understand the basic biology and epidemiology of this disease.

Literature Cited

1. Adams, J. F. 1921. Observations on wheat scab in Pennsylvania and its pathological histology. Phytopathology 11:115-125.
2. Andersen, A. L. 1948. The development of *Gibberella zeae* headblight of wheat. Phytopathology 38:595-611.
3. Arthur, J. C. 1891. Wheat scab. Indiana Agric. Exp. Stn. Bull. 36:129-138.
4. Ayers, J. E., Pennypacker, S. P., Nelson, P. E., and Pennypacker, B. W. 1975. Environmental factors associated with airborne ascospores of *Gibberella zeae* in corn and wheat fields. (Abstr.) Phytopathology 65:835.
5. Bai, G.-H., Shaner, G., and Ohm, H. 1991. Effect of moist period on response of wheat cultivars to infection by *Fusarium graminearum*. (Abstr.) Phytopathology 81:1145-1146.
6. Bai, G.-H., Shaner, G., and Ohm, H. 1993. Inheritance of resistance to *Fusarium graminearum* in eight wheat cultivars. (Abstr.) Phytopathology 83:1414.
7. Bai, G.-H., and Xiao, Q.-P. 1989. Genetic analysis on resistance to scab in six wheat cultivars. Acta Agric. Shanghai 5(4):17-23.
8. Bai, G.-H., Zhou, C.-F., Ge, Y.-F., Qian, C.-M., Chen, Z.-D., and Yao, G.-C. 1989. A study on scab-resistance in new wheat cultivars and advanced lines. Jiangsu Agric. Sci. 7:20-22.
9. Bai, G.-H., Zhou, C.-F., Qian, C.-M., Xia, S.-S., and Ge, Y.-F. 1989. An analysis on combining ability of resistance to scab and other characters in eight wheat cultivars. Jiangsu Agric. Sci., Suppl. 1:79-83.
10. Bai, G.-H., Zhou, C.-F., Qian, C.-M., Xia, S.-S., Ge, Y.-F., and Chen, Z.-D. 1990. Genetic analysis on resistant genes to scab extension in wheat cultivars. (In Chinese.) Pages 171-177 in: Advances in

Researches on Inheritance to Diseases in Major Crops. L.-H. Zhu, ed. Jiangsu Sci-Tech. Publishing House, Nanjing, China.

11. Baird, R. E. 1993. Effects of foliar fungicides on wheat, 1992. Fungic. Nematicide Tests. 48:223.

12. Bennett, F. T. 1931. *Gibberella saubinetii* (Mont) Sacc. on British cereals. II Physiological and pathological studies. Ann. Appl. Biol. 18:158-177.

13. Chen, C.-H. 1983. A study on the inheritance of scab-resistance in wheat. Acta Agric. Univ. Zhejiangensis 9(2):115-126.

14. Christensen, J. J., Stakman, E. C., and Immer, F. R. 1929. Susceptibility of wheat varieties and hybrids to fusarial head blight in Minnesota. Minn. Agric. Exp. Stn. Bull. 59.

15. Clear, R. M., and Abramson, D. 1986. Occurrence of *Fusarium* head blight and deoxynivalenol (vomitoxin) in two samples of Manitoba wheat in 1984. Can. Plant Dis. Surv. 66(1):9-11.

16. Francis, R. G., and Burgess, L. W. 1977. Characteristics of two populations of *Fusarium roseum 'Graminearum'* in eastern Australia. Trans. Br. Mycol. Soc. 68(3):421-427.

17. Hanson, E. W., Ausemus, E. R., and Stakman, E. C. 1950. Varietal resistance of spring wheats to Fusarial head blight. Phytopathology 40:902-914.

18. Hart, L. P., Pestka, J. J., and Liu, M. T. 1984. Effect of kernel development and wet periods on production of deoxynivalenol in wheat infected with *Gibberella zeae*. Phytopathology 74:1415-1418.

19. Helentjaris, T. G. 1992. RFLP analysis for manipulating agronomic traits in plants. Pages 357-372 in: Plant Breeding in 1990s. Proc. Symp. Plant Breed. 1990s. H. E. Stalker and J. P. Murphy, eds. North Carolina State University, Raleigh.

20. Lewellen, R. T., Sharp, E. L., and Hehn, E. R. 1967. Major and minor genes in wheat for resistance to *Puccinia striiformis* and their responses to temperature changes. Can. J. Bot. 45:2155-2172.

21. Li, Y.-F. and Yu, Y.-J. 1988. Monosomic analysis for scab resistance index in wheat cultivar "WZHHS." J. Huazhong Agric. Univ. 7(4):327-331.

22. Liao, Y.-C., and Yu, Y.-J. 1985. Genetic analysis on scab-resistance in local wheat cultivar Wang Shuibai. J. Huazhong Agric. Univ. 4(2):6-14.33.

23. Liu, D.-J., Weng, Y.-Q., Chen, P.-D., and Wang, Y.-N. 1989. Gene transfer of scab resistance from *Roegneria kamoji* and *Elymus giganteus* to common wheat. Jiangsu Agric. Sci. Suppl. 1:97-100.

24. Liu, Z.-Z., and Wang, Z.-Y. 1990. Improved scab-resistance in China: Sources of resistance and problems. Pages 178-188 in: Wheat for the Nontraditional Warm Areas. Proc. Int. Conf. D. A. Saunders, ed. CIMMYT, Mexico, D.F.

25. Liu, Z.-Z., Wang, Z.-Y., Huang, D.-C., Zhao, W.-J., Huang, X.-M., Yao, Q.-H., Sun, X.-J., and Yang, Y. M. 1991. Generality of scab-resistance transgression in wheat and utilization of scab-resistance genetic resources. Acta Agric. Shanghai 7:65-70.

26. Lu, W.-Z., Liu, F.-L., Lin, Y.-B., and Wang, C.-L. 1990. Correlation analysis between resistance to scab and plant height in wheat. Pages 229-234 in: Advances in Researches on Inheritance to Diseases in Major Crops. L.-H. Zhu, ed.

Jiangsu Sci-Tech. Publishing House, Nanjing, China.

27. Martin, R. A., and Johnston, H. W. 1982. Effects and control of *Fusarium* diseases of cereal grains in the Atlantic Provinces. Can. J. Plant Pathol. 4:210-216.

28. Mesterhazy, A. 1981. The role of aggressiveness of *Fusarium graminearum* isolates in the inoculation tests on wheat in seedling state. Acta Phytopathol. Acad. Sci. Hung. 16(3-4):281-292.

29. Mesterhazy, A. 1987. Selection of head blight resistant wheats through improved seedling resistance. Plant Breed. 98:23-36.

30. Miller, J. D., Young, J. C., and Arnison, P. G. 1986. Degradation of deoxynivalenol by suspension cultures of the *Fusarium* head blight resistant wheat cultivar Frontana. Can. J. Plant Pathol. 8:147-150.

31. Miller, J. D., Young, J. C., and Sampson, D. R. 1985. Deoxynivalenol and *Fusarium* head blight resistance in spring cereals. Phytopathol. Z. 113:359-367.

32. Miller, J. D., Young, J. C., and Trenholm, H. L. 1983. *Fusarium* toxins in field corn: I Parameters associated with fungal growth and production of deoxynivalenol and other mycotoxins. Can. J. Bot. 61:3080-3087.

33. Nakagawa, M. 1955. Study on the resistance of wheat varieties to *Gibberella saubinetii*. II Genetic factors affecting resistance to *Gibberella saubinettii*. Jpn. J. Breed. 5:15-22.

34. Nyvall, R. F. 1970. Chlamydospores of *Fusarium roseum* 'Graminearum' as survival structures. Phytopathology 60:1175-1177.

35. Oswald, J. W. 1949. Cultural variation, taxonomy and pathogenicity of *Fusarium* species associated with cereal root rots. Phytopathology 39:359-376.

36. Pugh, G. W., Johann, H., and Dickson, J. G. 1933. Factors affecting infection of wheat heads by *Gibberella saubinetii*. J. Agric. Res. 46:771-797.

37. Purss, G. S. 1971. Pathogenic specialization in *Fusarium graminearum* on wheat and corn. Aust. J. Agric. Res. 22:553-561.

38. Rafalski, J. A., and Tingey, S. V. 1993. Genetic diagnostics in plant breeding: RAPDs, Microsatellites and machines. Trends Genet. 9:275-280.

39. Reis, E. M. 1990. Integrated disease management-The changing concepts of controlling head blight and spot blotch. Pages 165-177 in: Wheat for the Nontraditional Warm Areas. Proc. Int. Conf. D. A. Saunders, ed. CIMMYT, Mexico, D.F.

40. Schroeder, H. W., and Christensen, J. J. 1963. Factors affecting resistance of wheat to scab caused by *Gibberella zeae*. Phytopathology 53:831-838.

41. Seaman, W. L. 1982. Epidemiology and control of mycotoxigenic fusaria on cereal grains. Can. J. Plant Pathol. 4:187-190.

42. Shaner, G., and Buechley, G. 1992. Effect of foliar fungicides on wheat, 1991. Fungic. Nematicide Tests. 47:206-207.

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43. Sheng, C.-S., and He, J.-M. 1989. Our experience with wheat breeding for scab resistance. Jiangsu Agric. Sci., Suppl. 1:58-59.

44. Snijders, C. H. A. 1990. The inheritance of resistance to head blight caused by *Fusarium culmorum* in winter wheat. Euphytica 50:11-18.

45. Snijders, C. H. A. 1990. Diallel analysis of resistance to head blight caused by *Fusarium culmorum* in winter wheat. Euphytica 50:1-9.

46. Snijders, C. H. A. 1990. Fusarium head blight and mycotoxin contamination of wheat, a review. Neth. J. Plant Pathol. 96:187-198.

47. Strange, R. N., Majer, J. R., and Smith, H. 1974. The isolation and identification of choline and betaine as the two major components in anthers and wheat germ that stimulate *Fusarium graminearum* in vitro. Physiol. Plant Pathol. 4:277-290.

48. Strange, R. N., and Smith, H. 1978. Specificity of choline and betaine as stimulants of *Fusarium graminearum*. Trans. Br. Mycol. Soc. 70:187-192.

49. Sutton, J. C. 1982. Epidemiology of wheat head blight and maize ear rot caused by *Fusarium graminearum*. Can. J. Plant Pathol. 4:195-209.

50. Teich, A. H., and Hamilton, J. R. 1985. Effect of cultural practices, soil phosphorous, potassium, and pH on the incidence of Fusarium head blight and deoxynivalenol levels in wheat. Appl. Environ. Microbiol. 49:1429-1431.

51. Teich, A. H., and Nelson, K. 1984. Survey of Fusarium head blight and possible effects of cultural practices in wheat fields in Lambton county in 1983. Can. Plant Dis. Surv. 64(1):11-13.

52. Tu, C. 1929. Physiologic specialization in *Fusarium* spp. causing headblight on small grains. Phytopathology 19:143-154.

53. Tuite, J., Shaner, G., and Everson, R. J. 1990. Wheat scab in soft red winter wheat in Indiana in 1986 and its relation to some quality measurements. Plant Dis. 74:959-962.

54. Wang, Y.-Z., Chen, H.-G., and Hao, S.-F. 1990. Interaction between wheat varieties and pathogenic isolates causing scab of wheat. Pages 206-210 in: Advances in Researches on Inheritance to Diseases in Major Crops. L.-H. Zhu, ed. Jiangsu Sci-Tech. Publishing House, Nanjing, China.

55. Wang, Y.-Z., and Miller, J. D. 1987. Screening techniques and sources of resistance to Fusarium head blight. Pages 239-250 in: Wheat Production Constraints in Tropical Environments. Proc. Int. Conf., 1987. CIMMYT, Mexico, D.F.

56. Wang, Y.-Z., Yong, X.-N., and Xiao, Q.-P. 1982. The improvement of identification technique of scab resistance of wheat and the development of resistant sources. Sci. Agric. Sin. 5:67-77.

57. Wang, Z.-Y., Liu, Z.-Z., Zhao, W.-J., Huang, D.-Z., and Huang, X.-M. 1989. Advance of scab resistance testing and improvement in wheat varieties. Jiangsu Agric. Sci., Suppl. 1:64-68.

58. Warren, H. L., and Kommedahl, T. 1973. Fertilization and wheat refuse effects on *Fusarium* species associated with wheat roots in Minnesota. Phytopathology 63:103-108.

59. Wiese, M. V. 1987. Scab (head blight). Pages 16-18 in: Compendium of Wheat Diseases. 2nd ed. American Phytopathological Society, St. Paul, MN.

60. Wu, Z.-S. 1986. Development of a gene pool with improved resistance to scab in wheat by using the dominant male sterile gene Ta 1. Proc. Int. Wheat Conf., 1986, Rabat, Morocco.

61. Xiao, Q.-P, Wu, Z.-F., and Pen, L.-X. 1989. Dynamic observation of interaction between *Fusarium graminearum* causing wheat scab and varieties with different types of resistance. Jiangsu Agric. Sci., Suppl. 1:69-71.

62. Xu, Y.-G., and Fan. Z.-D. 1985. Evaluation and differentiation of pathogenicity of *Fusarium graminearum* on wheat cultivars. Acta Phytopathol. Sin. 12(4):53-57.

63. Yang, Y.-Z. 1989. The theory and practice on breeding for scab resistance. Jiangsu Agric. Sci., Suppl. 1:60-63.

64. Yu, Y.-J. 1982. Monosomic analysis for scab resistance and yield components in the wheat cultivar Sumai 3. Cereal Res. Commun. 10(3-4):185-189.

65. Yu, Y.-J. 1990. Genetic analysis for scab resistance in four wheat cultivars, PHJZM, HHDTB, CYHM and YGFZ. Pages 197-205 in: Advances in Researches on Inheritance to Diseases in Major Crops. L.-H. Zhu, ed. Jiangsu Sci-Tech. Publishing House, Nanjing, China.

66. Zhang, L.-Q., Wang, Y.-Z., and Zhang, L. 1990. Advances of studies on genetics of resistance to scab and its pathogens (Review). Pages 165-170 in: Advances in Researches on Inheritance to Diseases in Major Crops. L.-H. Zhu, ed. Jiangsu Sci-Tech. Publishing House, Nanjing, China.

67. Zhou, C.-F. 1985. Production constraints and research priorities in the southern winter wheat region of China. Pages 72-77 in: Wheat for More Tropical Environments. CIMMYT, Mexico, D.F.

68. Zhou, M.-L., Bai, G.-H., Wang, S., and Zhao, Y.-H. 1988. Comparison of scab resistance among the wheat cultivars and their isogenic lines with different plant heights. Acta Agric. Jiangsu 4(1):25-30.

69. Zhu, H., and Fan, Y. 1989. Wheat scab epidemics and efficacy of testing for resistance in field in northern Fujiang Province. Jiangsu Agric. Sci., Suppl. 1:75-78.

70. Zhuang, Q.-S., and Li, Z.-S. 1993. Present status of wheat breeding and related genetic study in China. Wheat Inf. Serv. 76:1-15.